Université de Montréal
et du monde.

Not Your Typical Stroke(s)

A *Carotid* Case Presentation

2026 Winnipeg Vascular & Endovascular Symposium
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Disclosures

I have no disclosures.

Case Presentation

Patient – 70-year-old man originally from Lebanon

Past Medical History

- Diabetes mellitus (HbA1c 9.6%)
- Hypertension
- Benign prostatic hyperplasia
- Osteoporosis

Allergies –

Medication –

- Forxiga, diamicron, metformine
- Telmisartan, hydrochlorothiazide

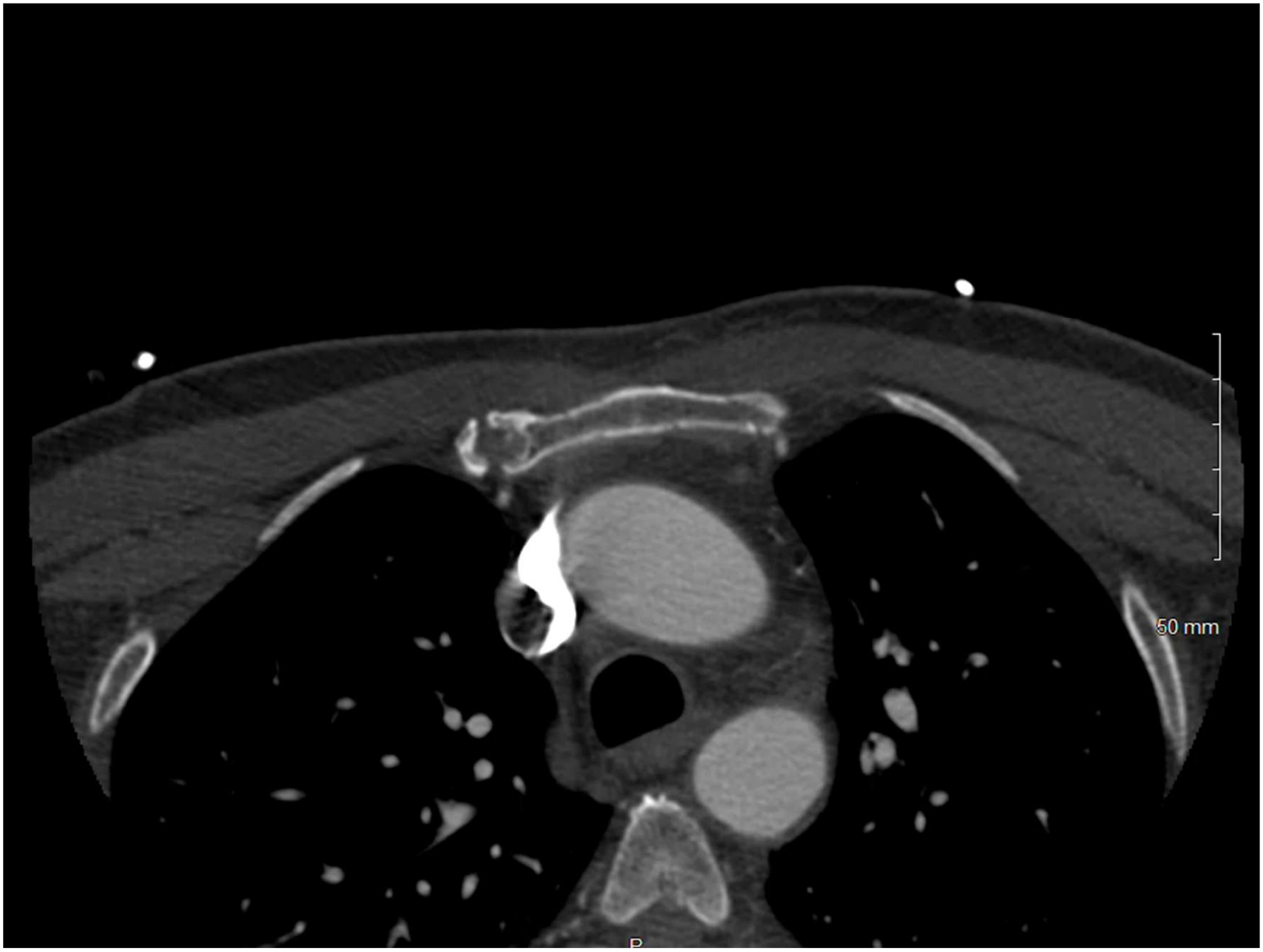
S – Presents to the ED for dizziness and language disturbance

- Last known well > 6 hours prior, woke up with symptoms
- History limited due to language barrier

O – Initial examination – Broca's aphasia, positive right pronator drift

Assessment – NIHSS 10, initial impression – Probable left middle cerebral artery (MCA) territory stroke

Initial CTA



Case Presentation

Reassessment in the stroke unit

- Speech disturbance: dysarthria vs aphasia
- Extraocular movements: abnormal, with leftward gaze preference
- Marked ataxia with dysmetria on finger-to-nose testing, left > right

- 48h later, **MRI** – Left pontine and medullary stroke, no infarcts in the MCA territory
- Final Dx – Vertebrobasilar stroke, secondary to intra-cranial vascular disease
  - TTE, Holter, ECG – Normal
  - CTA – extracranial and intracranial carotid atherosclerosis
- Hospitalization complicated with severe dysphagia and aspiration pneumonia

Management –

- Optimal medical therapy – ASA 80 mg PO OD, Lipitor 80 mg PO OD, antibiotics
- Feeding gastrostomy
- PT/OT
- Transfered to rehab after 10 days

Labs

- CBC N
- E+/Cr N
- CRP 26

2 Months Later

Readmission – Loss of autonomy and mobility; worsening balance

- CT – New findings suggesting vertebrobasilar infarcts (third episode)
- Plavix 75 mg DIE OD
- Lipitor –» Pravastatin (non-adherent with history of myalgia)
- OT / PT

10 days later – New-onset aphasia

CTA and Vascular consultation

On our evaluation –

- Known sequelae of vertebrobasilar strokes – Ataxia/dysmetria, dysarthria, dysphagia
- Aphasia reported by family

Labs –

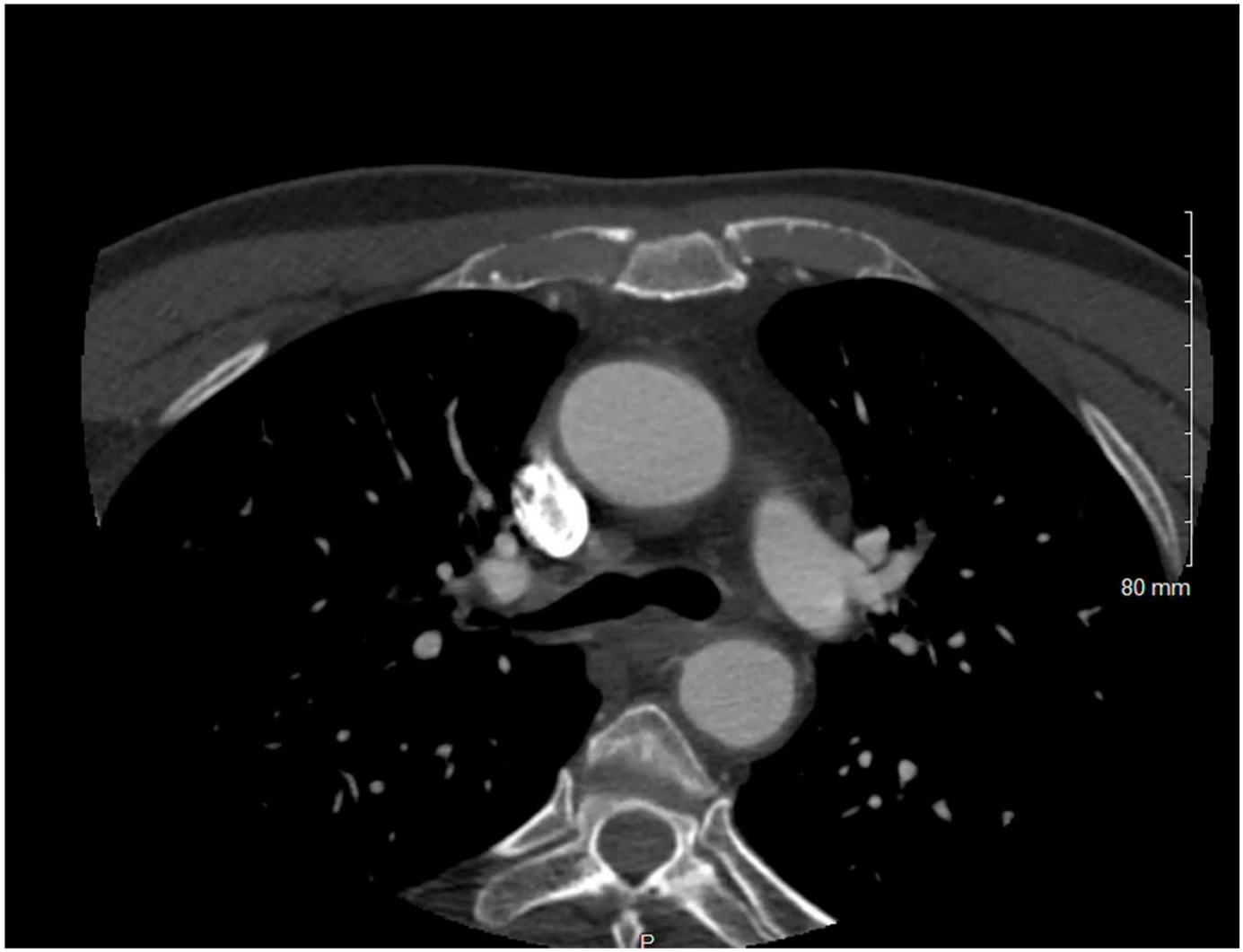
Hb 124 – WBC 7.3 – PLT 295

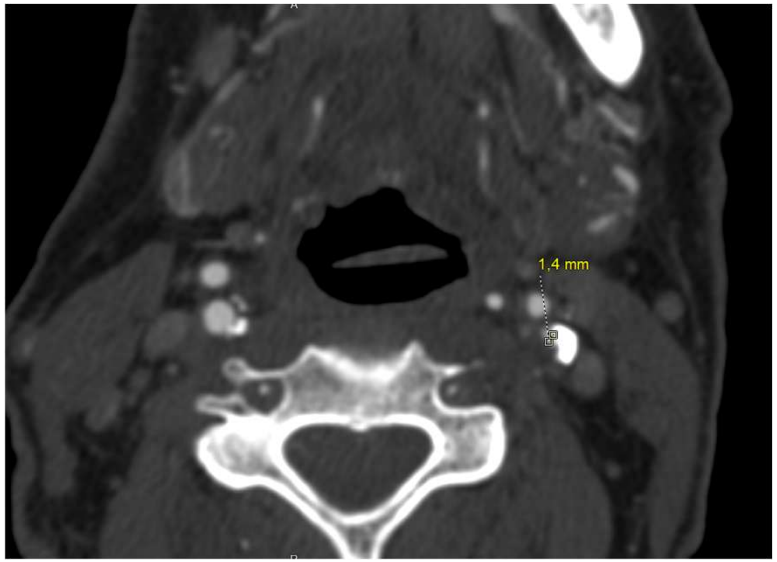
Cr 89, E+ N

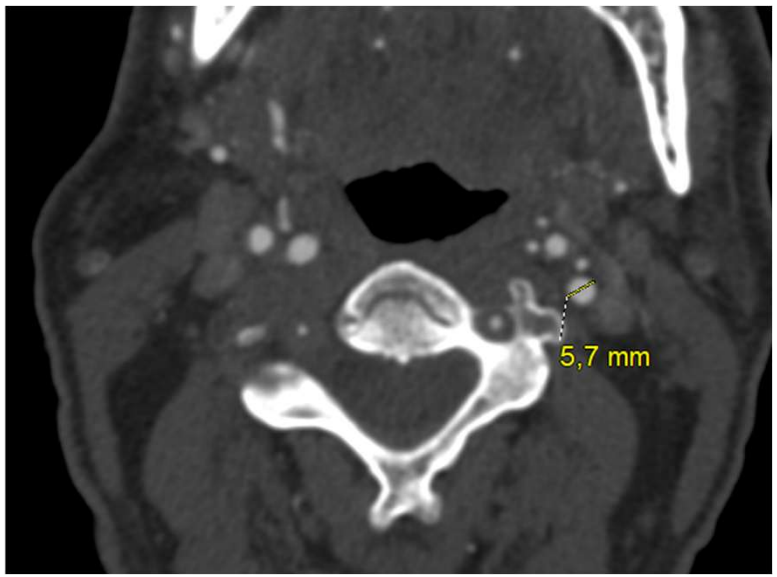
CRP 13

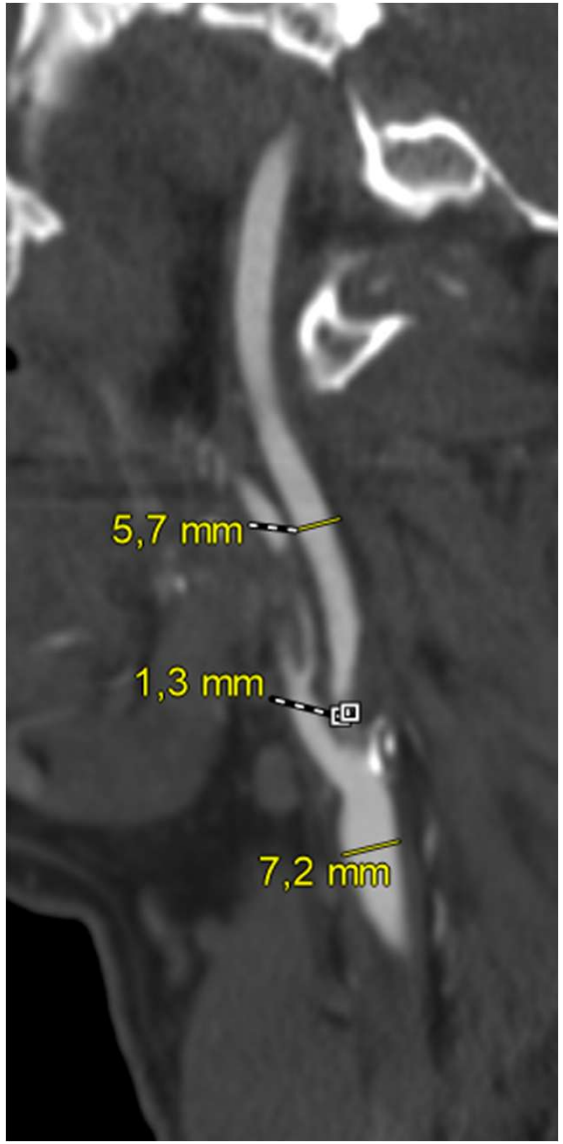
COVID +

CTA









Per NASCET criteria –

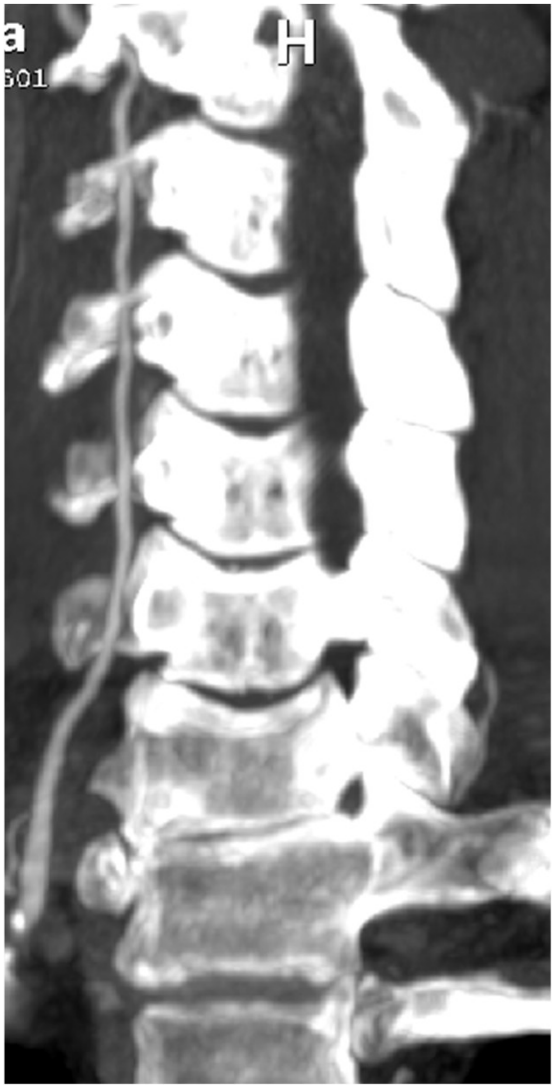
- Left internal carotid artery –

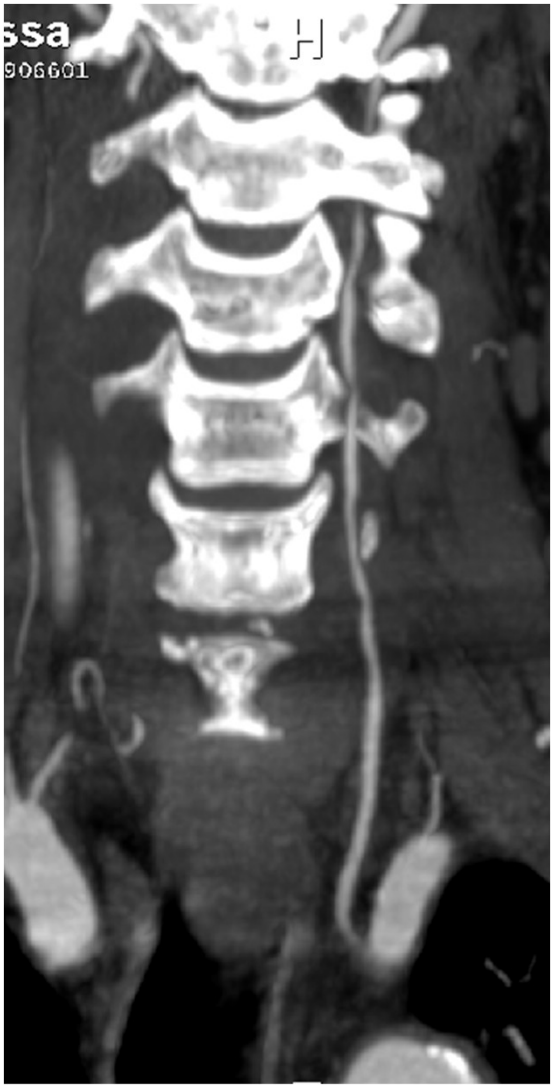
(A – B) / A x 100

(5.7 – 1.4) / 5.7 x 100 = 75%

With infarcts in the left MCA territory

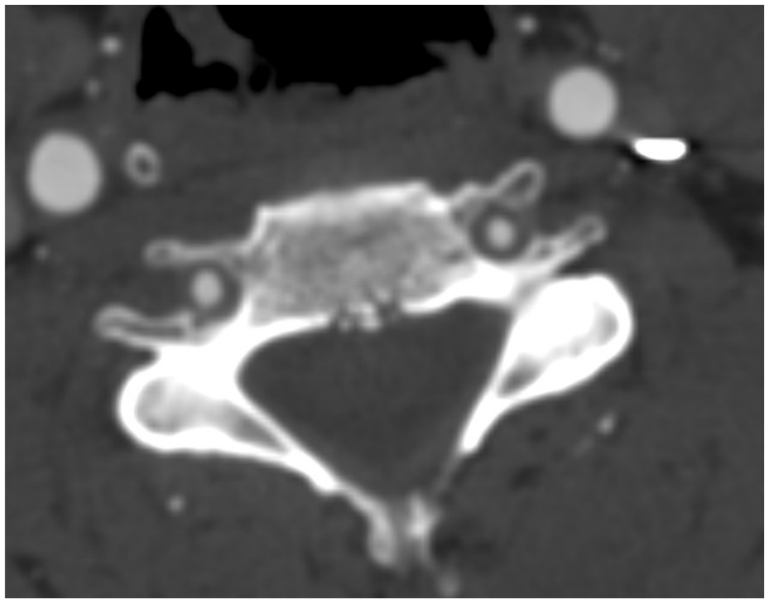
- Right ICA < 50%

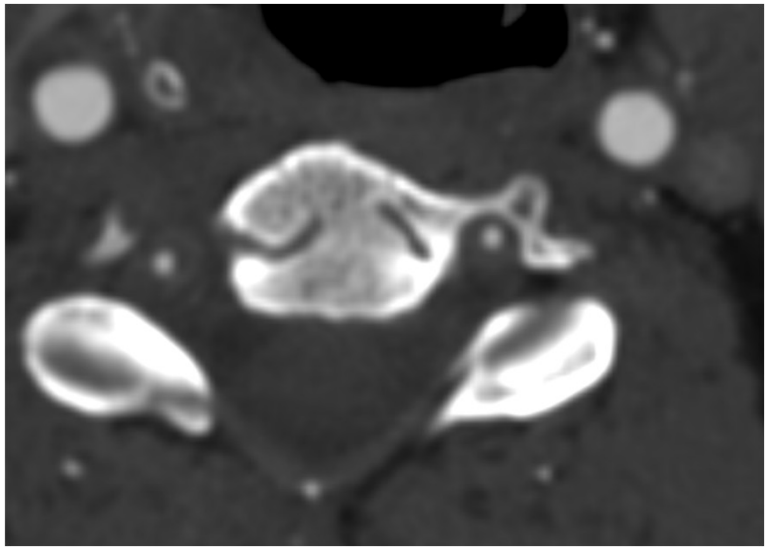


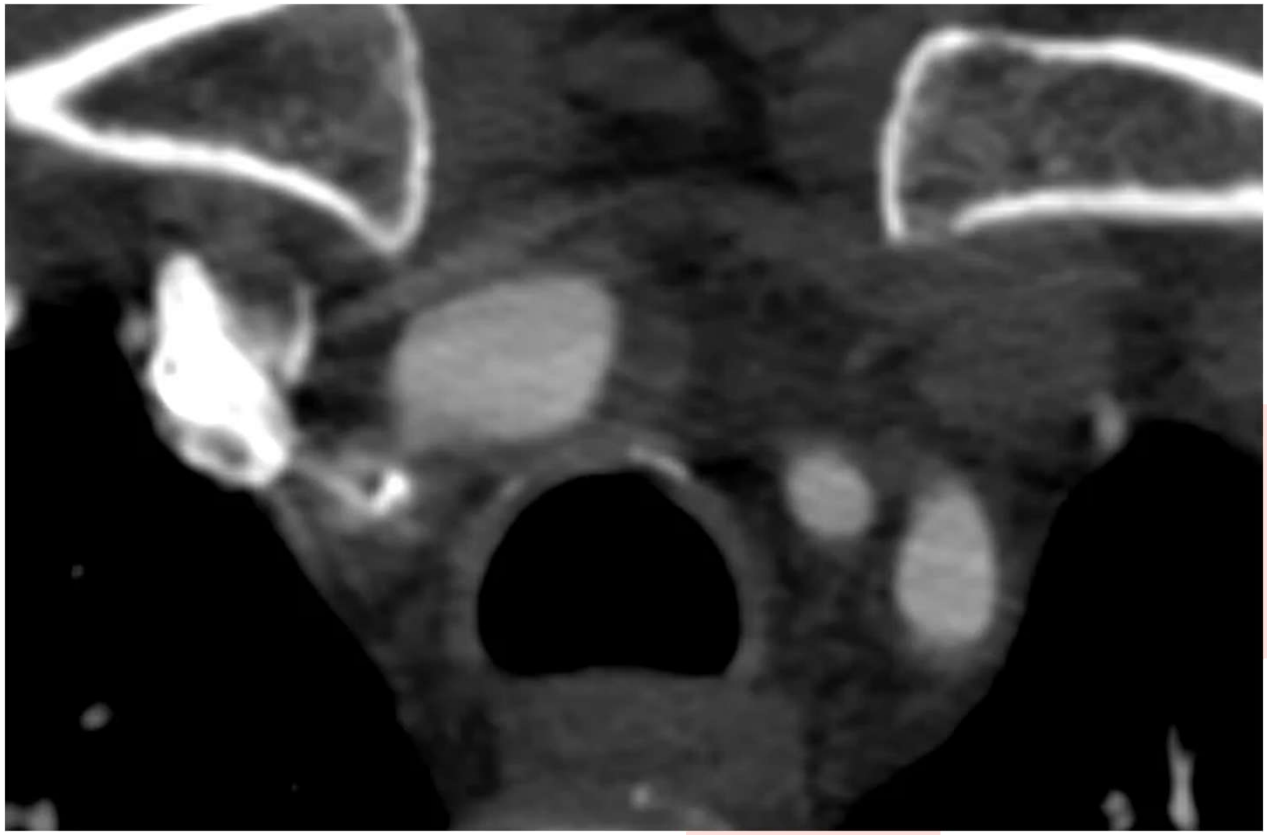


New Vascular Findings –

- Reduction in caliber of both vertebral arteries (V2 segments)
- Periarterial inflammation
- Bilateral focal occlusions (V3 and V4 segments)







To Summarize …

70-year-old man on optimal medical therapy

- (1) Status post vertebrobasilar strokes (3 episodes)
  - With inflammatory changes of vertebral arteries
  - Normal bloodwork; positive to SARS-CoV-2 with secondary elevation of CRP
  - Etiology of stroke – Intracranial disease

- (2) Status post left middle cerebral artery stroke with a 75% stenosis
  - With documented infarct on CT

Assessment – Left symptomatic severe carotid stenosis

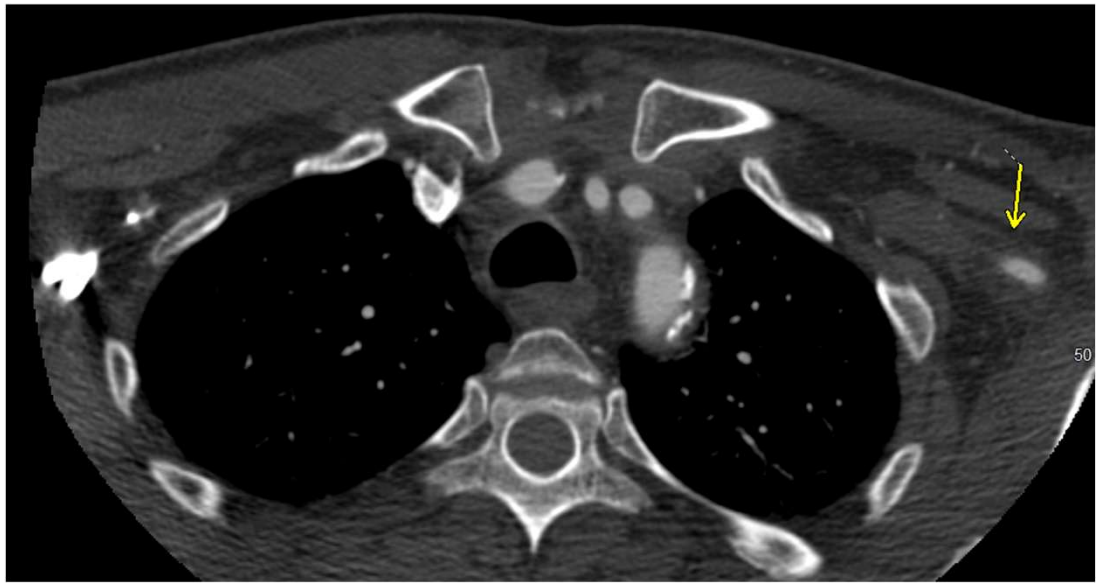
Plan – Left eversion carotid endarterectomy

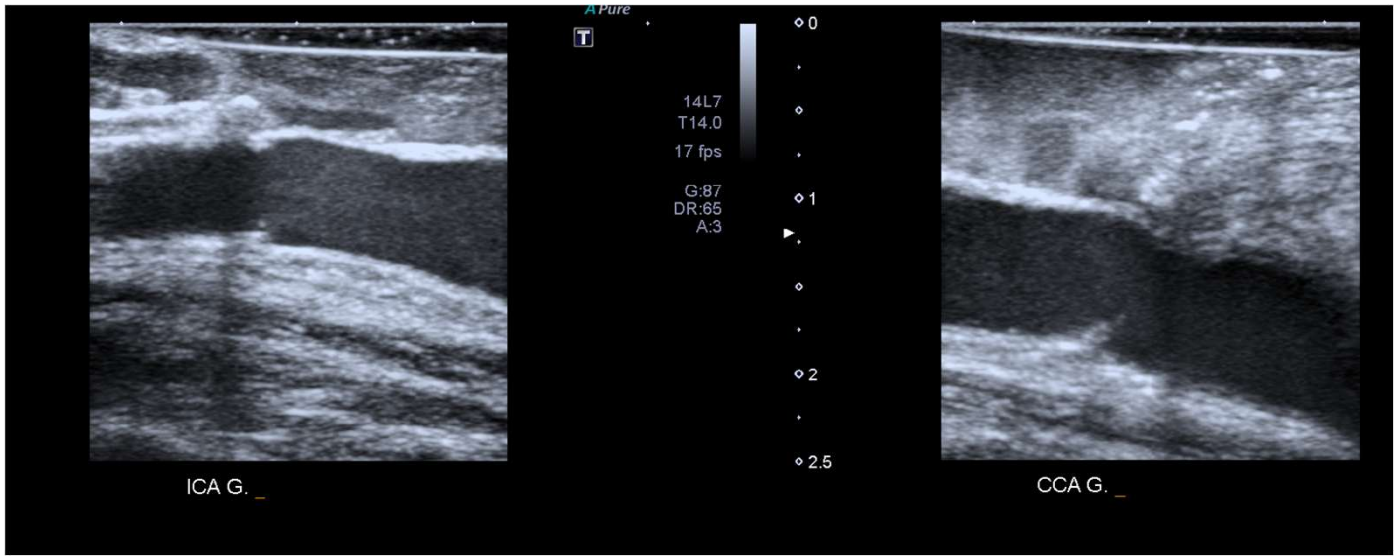
- Periarterial inflammation, no other significant findings, no complications

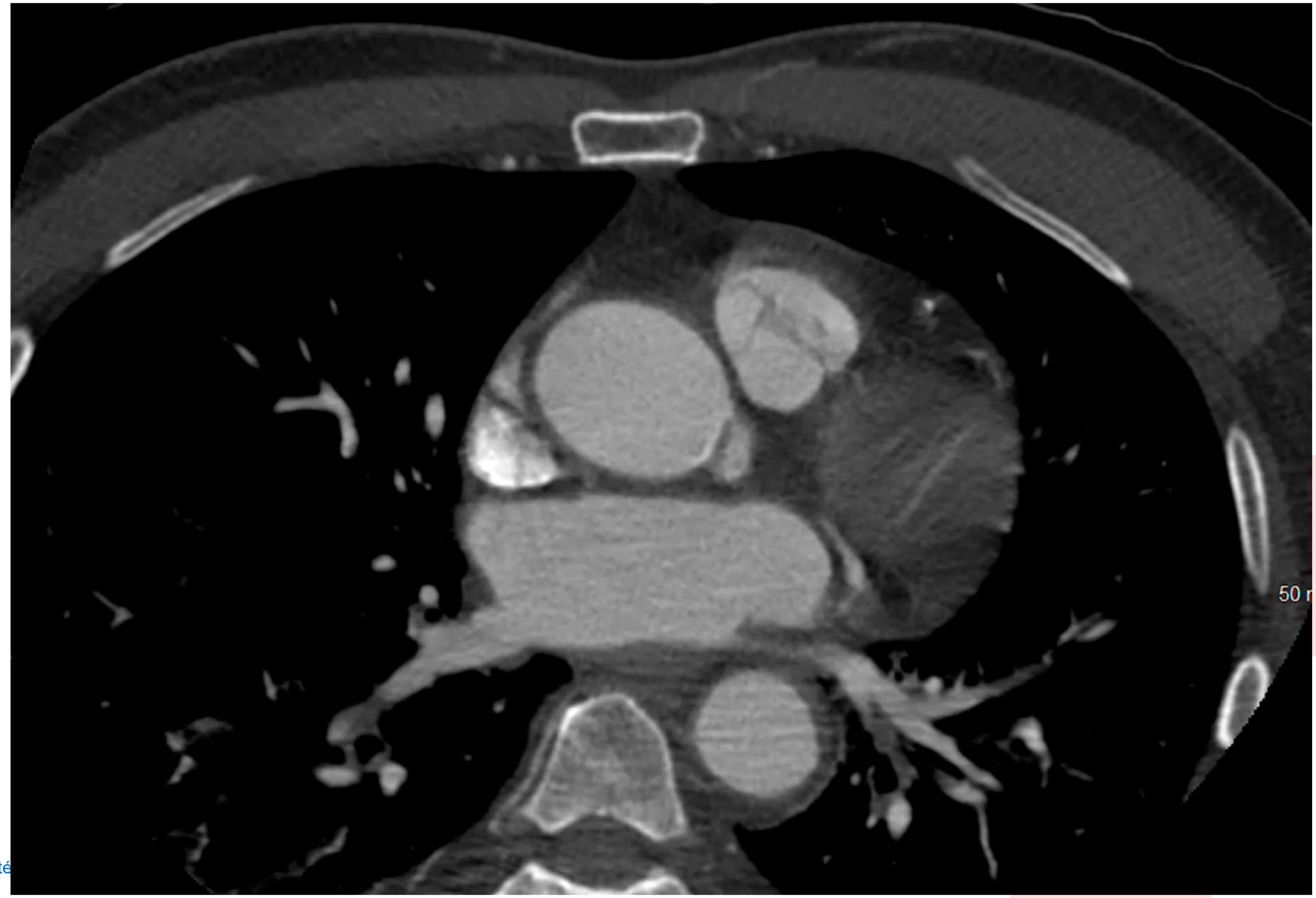
PO # 1

New on set right hemi-paresia – DUS / CTA

- No evidence of complications at the surgical site
- Inflammatory infiltrations of the verterbral arteries (as previously described)
- New signs of ischemic lesions in the left posterior circulation
- Pulmonary embolism






50 m

PO # 1

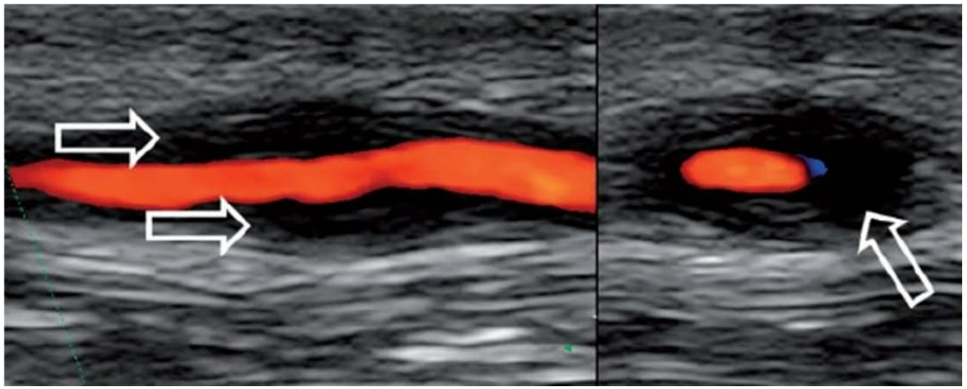
Internal medicine consultation –

- COVID most probable cause of PE (started on UFH)
- PFO – less probable (N TTE), SAPL ?, no evidence of active cancer, suggestive of GCA… ?
- **Temporal artery ultrasound – Significantly abnormal**
  - *Hypoechoic wall thicknening (halo sign)*
  - *Non-compressible arteries*
  - *Lumen stenosis ou occlusions (turbulent flot, absence of signal)*

Assessment – Giant Cell Arteritis with severe Steno-occlusive disease

Plan –

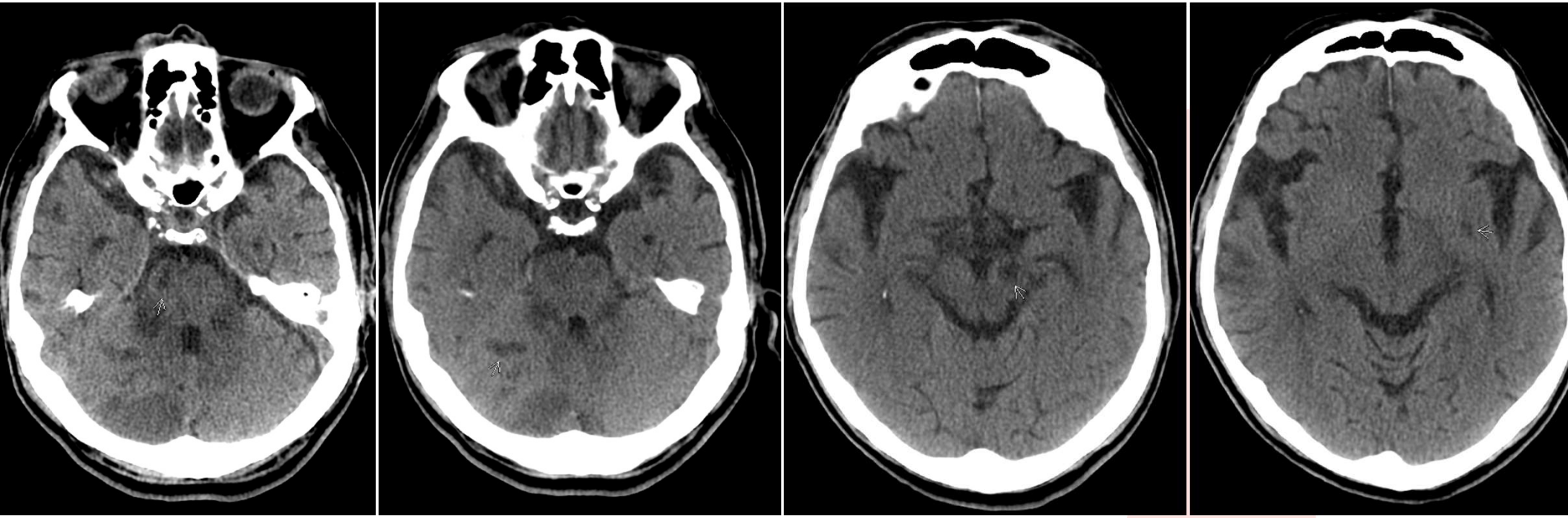
- Solumedrol (methylprenisolone) 500 IV DIE x 3 jours
- Tocilizumab (actemra) 530mg IV x 1 dose (8 mg/kg)
  Consider Methotrexate afterwards
- Prednisone 80 mg PO DIE x 7 days, then step down



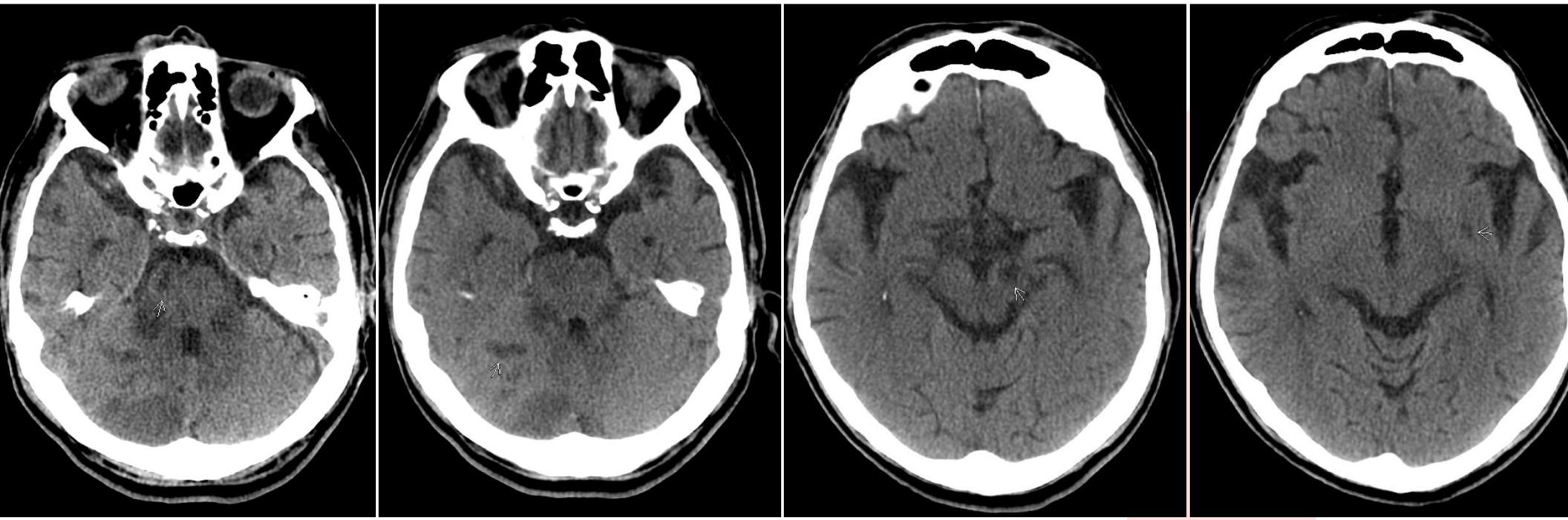
Halo sign in Giant Cell Arteritis (Schmidt 2018)

Evolution – Over the next 10 days

- Aspiration pneumonia, lower GI bleed, admission to ICU
- Progression with new ischemic lesions bilateraly in the PICA territory, midline shifts



What could have been done differently and what can we learn ?

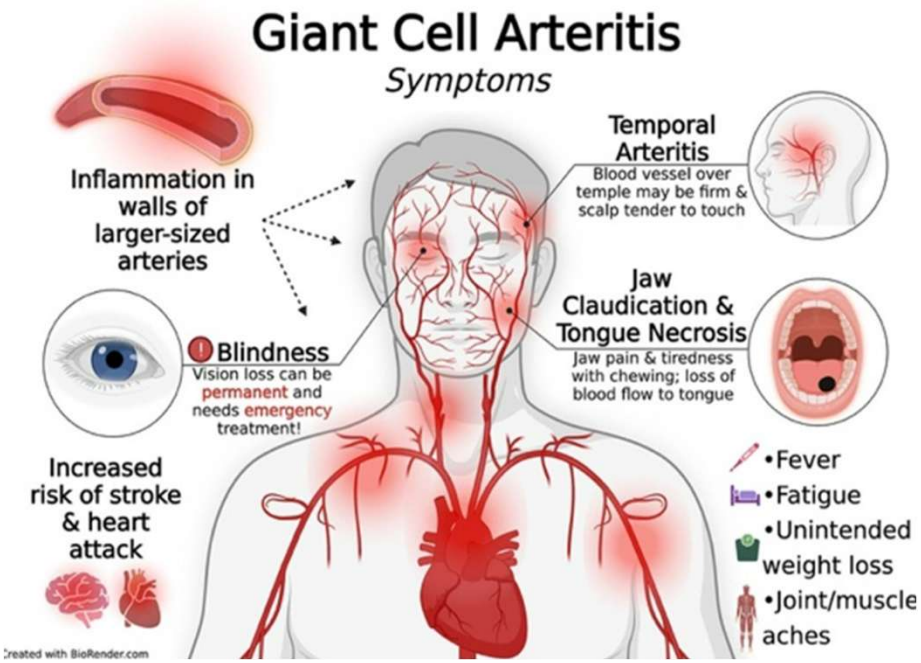


Overview of Giant Cell Arteritis

Definition – Large and medium vessel vasculitis primarily affecting patients of > 50 years, the exact cause remains unknown

Presentation –

- New-onset headache with evidence of systemic inflammation
- Cranial symptoms – Scalp tenderness, diplopia, bindness/visual loss,
- Will rarely presente with stroke/TIA/amaurosis fugax



Overview of Giant Cell Arteritis

Definition – 3/5 criteria of the ACR

- Temporal biopsie remains the gold standard
  - Intimal fibrosis
  - Medial scarring
  - Asymetrical destruction of the internal elastic lamina
- Imaging to assess large vessel involvement

TABLE 138.2

American College of Rheumatology Criteria for Diagnosis of Giant Cell Arteritis (1990)

Criterion	Definition
Age at disease onset >50 years	Development of symptoms or findings beginning at age 50 or older
New headache	New onset headache
Temporal artery abnormality	Temporal artery tenderness to palpation or decreased pulsation without evidence of arteriosclerosis of cervical arteries
Elevated erythrocyte sedimentation rate (ESR)	ESR >50 mm/h by Westergren method
Abnormal artery biopsy	Biopsy specimen showing vasculitis characterized by predominance of mononuclear cell infiltration or granulomatous inflammation, usually with multinucleated giant cells

To meet criteria for a diagnosis of giant cell arteritis, at least three out of the five criteria should be present. [17]

GCA and Strokes – Review of Literature

- Rare cause of stroke, but the most common vasculitis in the elderly – Peak at 70 to 80
- Often occult GCA presentation with rare occurrence of typical cranial features
- Will complicate 2.8 to 7% to GCA cases, most commonly within 1 month of diagnosis and initiation of treatment
  - Patients typically older – median age of 78 to 85 years old
  - Risk factors of having a stroke with proven GCA – Male, vision loss, hypertension, smoking,

Stroke characteristics – Ischemic (watershed, embolic), VB 75%

- Multiple stenosis effecting segments V2 to V4
- Might represent 0.15% to 0.4% of all patients admitted to the hospital with a stroke and
- Might represent 2.0% to 3.1% of VB strokes admitted to the hospital

Evolution – High recurrence rate despite optimal medical treatment, with high mortality (up to 30%)

GCA and Strokes – Review of Literature

Should we have screened this patient base on stroke territory ?

Red flags for a concomitant giant cell arteritis in patients with vertebrobasilar stroke: a cross-sectional study and systematic review

Ahmed Mohamed Elhfnawy[1,2,3] · Doaa Elsalamawy[2] · Mervat Abdelraouf[2] · Mira Schliesser[1] · Jens Volkmann[1] · Felix Fluri[1]

Prospective study –

- Screening of all patients admitted to the stroke unit with VB-stroke
  - DUS of vertebral and temporal arteries
  - CBC – ESR – CRP

Conclusion / risk factors of having GCA when presenting with VB stroke – Anemia, elevated inflammatory markers, multiple stenoses/occlusions in the VB territory, older age

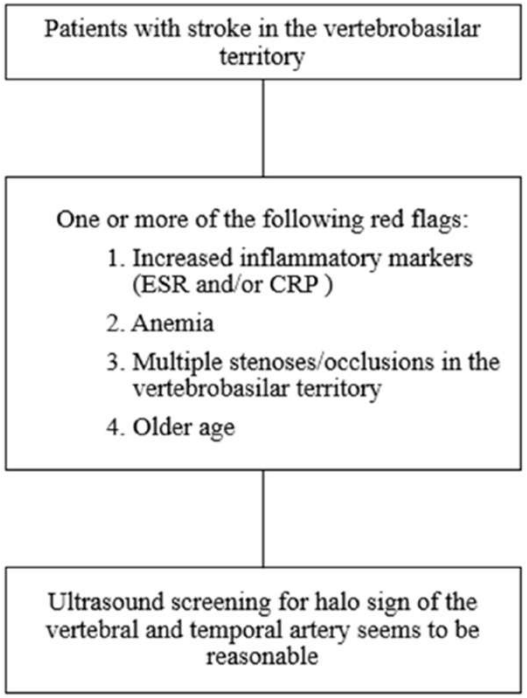


Fig. 2 Proposed flow chart showing the red flags raising suspicion and warranting screeing for giant cell arteritis among patients with vertebrobasilar stroke

Overview of Giant Cell Arteritis

Treatment strategies

Treatment –

- Corticosteroid therapy
  - Prednisone 40 à 60 mg PO DIE, or loading with methylprednisolone if severe presentation
- Anti-IL6 anti-body – For severe cases
- ASA with flow limiting stenosis or occlusions of the vertebral and carotid arteries

Surgical intervention		
Ungraded position statement: For any patient requiring surgical vascular intervention for GCA, the type and timing of intervention should be a collaborative decision between the vascular surgeon and rheumatologist.	‡	‡
Recommendation: For patients with severe GCA and worsening signs of limb/organ ischemia who are receiving immunosuppressive therapy, we conditionally recommend escalating immunosuppressive therapy over surgical intervention with escalation of immunosuppressive therapy.	24	Very low to low
Recommendation: For patients with GCA undergoing vascular surgical intervention, we conditionally recommend the use of high-dose GCs during the periprocedural period, if the patient has active disease.	27	Very low

Overview of Giant Cell Arteritis

Treatment strategies and the role of surgical interventions

Intracranial Internal Carotid Artery Angioplasthy and Stenting in Giant Cell Arteritis

Antonio Méndez Guerrero, Fernando Sierra-Hidalgo, Patricia Calleja, Pedro Navia, Jorge Campollo, Jaime Díaz-Guzmán

From the Stroke Unit, Department of Neurology, Hospital Universitario 12 de Octubre, Madrid, Spain (AMG, FS-H, PC, JD-G); Instituto de Investigación Hospital 12 de Octubre (i+12), Madrid, Spain (FS-H); and Interventional Neuroradiology Unit, Hospital Universitario 12 de Octubre, Madrid, Spain (PN, JC).

Angioplastie carotidienne bilatérale chez une patiente avec infarctus cérébral sur maladie de Horton

Bilateral intracerebral angioplasty in a patient with stroke caused by giant cell arteritis

N. Chausson [a,], S. Olindo [a], A. Signaté [a], P. Cohen-Ténoudji [b], M. Aveillan [c], M. Saint-Vil [a], D. Smadja [a]*

« They were last chance therapy given the lack of response »

Take Home Messages

Dual pathology !

GCA – Common vasculitis, uncommon cause of stroke

- Consider in – VB stroke, older age, inflammatory markers (CRP, ESR, anemia) and multiple occlusions of vertebral arteries

Treatment– Medical with glucocorticoïde, could have a place for surgical interventions in select refractory cases as a salvage option, prognosis remains somber

"This case challenges us to look beyond luminal stenosis and recognize when the vessel wall itself is the disease."

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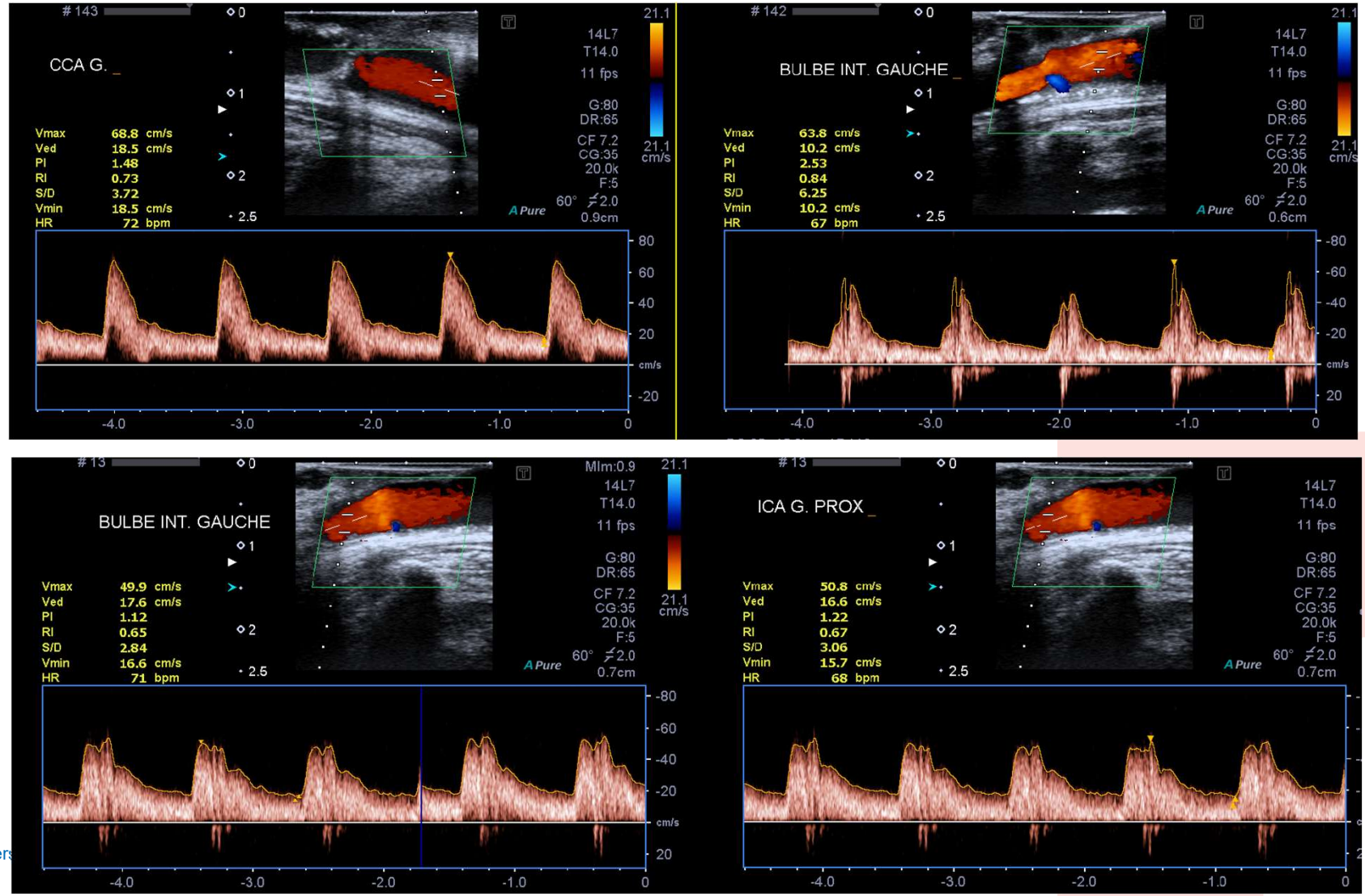
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Vmax 68.8 cm/s
Ved 18.5 cm/s
PI 1.48
RI 0.73
S/D 3.72
Vmin 18.5 cm/s
HR 72 bpm
BULBE INT. GAUCHE
Vmax 63.8 cm/s
Ved 10.2 cm/s
PI 2.53
RI 0.84
S/D 6.25
Vmin 10.2 cm/s
HR 67 bpm
BULBE INT. GAUCHE
Vmax 49.9 cm/s
Ved 17.6 cm/s
PI 1.12
RI 0.65
S/D 2.84
Vmin 16.6 cm/s
HR 71 bpm
ICA G. PROX
Vmax 50.8 cm/s
Ved 16.6 cm/s
PI 1.22
RI 0.67
S/D 3.06
Vmin 15.7 cm/s
HR 68 bpm
14L7
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CF 7.2
CG:35
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cm/s

GCA and Strokes – Review of Litterature

Increased incidence of giant cell arteritis and associated stroke during the COVID-19 pandemic in Spain: A nation-wide population study

Víctor Moreno-Torres [a,b,*], Vicente Soriano [a], Jorge Calderón-Parra [c], María Martínez-Urbistondo [b], Ana Treviño [a], Zayrho de San Vicente [d,e], Carmen de Mendoza [c,f], Guillermo Ruiz-Irastorza [g,h]

Results – Post-Pandemic – More admissions for GCA, and more admissions for GCA attributed stroke

Discussion – admissions Pathophysiology of GCA uncertain, reports of association with infections disease and other autoimmune or rheumatic diseases

Table 2
Epidemiology of GCA and GCA-associated stroke between 2016 and 2021 in Spain.

	2016 (*N* = 3,895,317)	2017 (*N* = 3,972,586)	2018 (*N* = 4,026,251)	2019 (N = 3,971,324)	2020 (N = 3,516,107)	2021 (N = 3,706,163)
Admissions in patients with GCA						
N	2757	2844	2822	3052	2788	2.995
Incidence per 100,000 admissions	70.1	71.6	70.1	76.9	79.3	80.8
Admissions with GCA-associated stroke						
N (%)	214 (7.8)	219 (7.7)	242 (8.6)	233 (7.6)	285 (10.2)	284 (9.5)
Incidence per 100,000 admissions	5.5	5.5	6	5.9	8.1	7.7